Abiotic predictors of avian botulism outbreaks in Utah

Scott C. Barras and John A. Kadlec

Abstract Avian botulism is a toxicosis responsible for large outbreaks in wild waterfowl and wading birds. The toxin is produced by the bacterium *Clostridium botulinum* type C. We gathered existing climatological and streamflow data for the Bear River Delta of northeastern Utah to determine effects of environmental factors on probability of occurrence of an avian botulism outbreak. We built a logistic regression model using weather variables and streamflow amounts for 28 years, with a response variable if an outbreak occurred during each year. Winter-summer precipitation amounts and summer streamflow were predictors of outbreak probability ($P<0.001$). Outbreaks were more likely to occur during years of high winter-summer precipitation and high Bear River flows during summer. We concluded that these outbreak predictors may contribute to water-level fluctuations in managed wetland units, associated previously with outbreaks in this and other systems.

Key words avian botulism, avian disease, *Clostridium botulinum* type C, waterfowl, wading birds, wetlands

Avian botulism is responsible for massive local mortalities of wetland birds, primarily in the western United States and Canada (Jensen and Price 1987). The disease is caused by consuming toxin produced by the bacterium *Clostridium botulinum* type C. The causative agent is an obligate anaerobe with a fermentative metabolism and is common in wetland sediments (Mitchell and Rosendal 1987, Sandler et al. 1993). Toxin is produced in actively growing bacteria that are infected by a bacteriophage containing the genetic code for toxin production (Eklund et al. 1971, 1987; Eklund and Poysky 1974). Species most affected include American avocets (*Recurvirostra americana*), green-winged teal (*Anas crecca carolinensis*), black-necked stilts (*Himantopus mexicanus*), and northern pintails (*Anas acuta*, Rosen 1971).

The exact mechanism leading to outbreak initiation is unknown, but it may be triggered by environmental conditions. Several mechanisms have been proposed for outbreak initiation, including the sludge-bed hypothesis and micro-environment concept (Enright 1971). The sludge-bed concept holds that decaying organic matter, mostly plants, produces the anaerobic environmental conditions necessary for *C. botulinum* to grow and produce toxin. In contrast, the micro-environment concept holds that the general ambient environment is irrelevant and that the environment within invertebrate or vertebrate carcasses stimulates the production of *C. botulinum* type C toxin (Bell et al. 1955).

Although outbreaks have been considered unpredictable (Rocke et al. 1999), decades of observations have associated major outbreaks with fluctuating water levels, high temperatures, brackish or saline marshes, and presence of dead animal carcasses (invertebrate or vertebrate; Sperry 1947, Hunter 1970, Duncan and Jensen 1976, Jensen and Price

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1987). Recent data suggest that living invertebrates also may play a role in toxin transfer (Rocke et al. 1999). Rosen (1971) observed that outbreaks occur during summer following winters with heavy precipitation. However, summer weather events such as high winds and rainfall immediately preceding outbreaks also have been associated with outbreak initiation (Coburn and Quartrup 1938). These events may contribute to fluctuating water levels in wetlands and flooding of mudflats, conditions generally associated with outbreaks.

Outbreaks are believed to grow in size from a positive feedback system called the maggot cycle. This process involves toxin production in vertebrate carcasses, consumption of toxin by fly larvae feeding on these carcasses, consumption of toxin-containing larvae by birds, and mortality of these birds (Hunter 1970, Locke and Friend 1989, Reed and Rocke 1992). Much evidence demonstrated toxin production in animal carcasses, as well as toxicity of fly maggots feeding on such carcasses (Bell et al. 1955, Lee et al. 1962, Hunter 1970, Reed and Rocke 1992). This mechanism also may account for many of the spatial and temporal mortality patterns observed in wetlands during botulism outbreaks.

The Great Salt Lake (GSL) marshes of Utah are important staging areas for migrating shorebirds and waterfowl and are sites of intensive management to ensure availability of high-quality habitats for the migrating birds that use the area (Kadlec and Smith 1989). However, severe avian botulism outbreaks are common in marshes along the eastern shore of the GSL (Rosen 1971) and have been recorded since 1915 (Wetmore 1915). These marshes were the site of a major outbreak in 1997 when approximately 400,000 birds died on the Bear River Delta (89,000 confirmed; United States Fish and Wildlife Service [USFWS], unpublished report). On the Bear River Migratory Bird Refuge (BRMBR) near Brigham City, Utah, long-term average mortality due to botulism has exceeded hunting mortality since 1950 (USFWS, unpublished report).

We compiled historical data on avian botulism mortality and environmental variables to determine whether abiotic variables were reliable predictors of outbreak frequency. We compared the results of these analyses to existing hypotheses describing outbreak initiation mechanisms and habitat features of the study sites to determine which factors may contribute to outbreak initiation.

Methods

Study area

Our study area was the Bear River Migratory Bird Refuge (N 41°28' 47.03",W112°16' 02.91", 1,285-m elevation) in northern Utah. The refuge consists of 16,600 ha of freshwater marshes constructed on the delta of the Bear River, which flows into the Bear River Bay of the GSL. BRMBR is approximately 15 km west of Brigham City, Utah. Average annual precipitation is 40.13 cm on the refuge. However, the primary source of water for the Bear River is snowmelt from the mountains of northeastern Utah, southwestern Wyoming, and southeastern Idaho. The refuge was inundated by salt water when the GSL flooded in 1984–86. Since the flood, dike reconstruction has subdivided the refuge into 8 large impoundments (average area approximately 2,000 ha each). Further subdivision of these impoundments will result in 20 smaller impoundments.

Dataset construction

Annual losses to avian botulism have been estimated by USFWS personnel responsible for monitoring impacts of the disease each year. We collected these data from refuge reports and personal communications with refuge staff. Total estimated avian losses were used in analyses. Annual necropsies of a sample of birds were conducted by USFWS personnel of the Avian Botulism Laboratory at Brigham City, Utah, and the National Wildlife Health Laboratory in Madison, Wisconsin, to officially document avian botulism as cause of death. Botulism mortality is an annual event in the GSL marshes that results in losses of from hundreds to hundreds of thousands of birds each year over a large, remote wetland area. We selected the threshold mortality level in excess of 5,000 birds to define outbreak status for purposes of binary classification of outbreak occurrence. We determined that this level best differentiated between years of routine annual mortality and years when the outbreaks were considered catastrophic. Our approach was to identify abiotic variables that predicted outbreaks sufficiently large to impose catastrophic impacts on local and migratory bird populations and the operational resources of local wildlife management agencies.

We compiled historical data on weather conditions (including temperature, precipitation, and wind speed), Bear River flow, GSL elevation, and

annual outbreak magnitude for BRMBR. The primary dataset included data for 28 years prior to the GSL flood in which all data were collected. Although avian botulism in Bear River marshes has been studied for more than 28 years, we were able to identify only 28 years prior to the GSL flood for which streamflow, climatological, and mortality data were available. We constructed a second dataset of post-flood data to validate predictions of models built with the first dataset. Data describing weather conditions were collected by the National Weather Service (NWS) station at nearby Corinne, Utah. We converted daily temperature, precipitation, and wind speed values to monthly and seasonal averages and totals. We used daily Bear River flow data collected from the United States Geological Service (USGS) station at Corinne. We converted daily flows to monthly and seasonal totals in cubic meters. We obtained GSL elevations from USGS monitoring stations.

Statistical analyses

We used stepwise logistic regression (SAS 1989) to determine which environmental variables (temperature [maximum and mean seasonal, annual], precipitation [seasonal and annual], wind speed, Bear River flow [seasonal and annual], GSL elevation, and waterfowl use days) best predicted botulism outbreaks in the BRMBR system. The model produced annual probabilities for outbreak occurrence. Probabilities ≥ 0.5 were deemed positive predictions, whereas those <0.5 were negative. We used a jackknife procedure for statistical validation of the best model (Mosteller and Tukey 1977). For further validation, we used the best model from the original dataset to predict outbreak occurrence for each of 7 years in the post-GSL flood dataset. We used 2-sample *t*-tests to illustrate differences in predictive variables during outbreak and non-outbreak years. We declared $\alpha = 0.05$, a priori.

Results

Stepwise logistic regression detected a significant predictive relationship (-2 Log Likelihood = 22.629, $\chi^2_2 = 16.044$, $P < 0.001$) for a model with winter-summer precipitation and summer stream flow as predictors ($\chi^2_1 = 4.368$, $P = 0.04$, odds ratio = 1.769 and $\chi^2_1 = 4.105$, $P = 0.04$, odds ratio = 1.013, respectively). The model fit was excellent as gauged by the Hosmer-Lemeshow goodness-of-fit test (Hosmer and Lemeshow 1989, $C_7 = 3.850$, $P = 0.80$). Outbreaks were predicted in years with high precipitation and summer flows. Jackknife validation found positive predictions correct in 9 of 13 years and negative predictions correct in 12 of 15 years (21 of 28 years, overall). Outbreaks occurred in 8 of 9 years when outbreak probabilities were >60%. Predictions of post-flood outbreaks were correct in 5 of 8 years.

Table 1. Weather and flow variables in botulism outbreak versus non-outbreak years at Corinne, Utah, 1950–1957 and 1964–1983.

Variable	Outbreak	Non-outbreak
Summer flow[a]	276,464A	110,016B
Winter-spring precipitation[b]	40.8A	33.7B
Mean seasonal precipitation[b]		
Spring	12.2A	12.2A
Summer	7.9A	7.0A
Mean daily temperature		
Spring	10.2A	10.1A
Summer	15.6A	15.9A

[a] Flow (m^3) of Bear River measured at Corinne, Utah, 1 June–31 August, 1950–1957, 1964–1983. Means within rows followed by the same letter do not differ ($P > 0.05$).

[b] Precipitation (cm) measured at Corinne, Utah, 1 November–31 August, 1950–1957, 1964–1983.

Winter-spring precipitation was greater ($t_{34} = -2.532$, $P = 0.02$) during outbreak years than non-outbreak years (Table 1). Summer Bear River flows also were greater ($t_{17.7} = -2.883$, $P = 0.01$) during outbreak years. Temperatures did not differ among outbreak and non-outbreak years during spring ($t_{34} = -0.251$, $P = 0.80$) or summer ($t_{33} = 0.747$, $P = 0.46$, Table 1). Spring and summer precipitation (Table 1) also were similar for outbreak and non-outbreak years ($t_{34} = -0.019$, $P = 0.94$, $t_{33} = -0.596$, $P = 0.56$, respectively).

We found no relationship between waterfowl abundance and outbreak frequency. The stepwise procedure did not include waterfowl use days (USFWS, unpublished report) calculated for BRMBR as a significant factor in the regression model. Additionally, we found no correlative relationship between waterfowl use days and outbreak magnitude ($r = -0.172$, $P = 0.41$) in a 25-year dataset, and waterfowl use days among outbreak and non-outbreak years did not differ ($t_{23} = 0.957$, $P = 0.35$).

Discussion

Outbreak triggers

Our analyses supported anecdotal observations (Hunter 1970, Rosen 1971) that heavy over-winter

precipitation increases the probability of avian botulism outbreaks in downstream wetlands. Although summer temperatures (Graham 1978) and precipitation events (Coburn and Quartrup 1938) also have been associated with outbreak initiation, conditions prior to summer months were the best predictors of outbreak probabilities in this study. This point was further emphasized by the lack of differences in spring and summer temperatures and precipitation among outbreak and non-outbreak years. Our winter–summer precipitation variable was an index of expected water available for runoff the following summer, whereas summer flow was a more direct measure of disturbance during the time of year that conditions are more optimal for growth of *Clostridia*. Thus, we propose that heavy winter–summer precipitation and summer flows may contribute to botulism outbreak frequency through fluctuating water levels and reflooding of mudflats during the period of active clostridial growth, events associated previously with outbreaks of avian botulism (Jensen and Williams 1964).

Our results do not dispel either existing hypothesized mechanism for outbreak initiation. Instead, increased precipitation and flows represent environmental stimuli that could initiate an outbreak under either hypothesized scenario. For example, increases in allochthanous inputs of dead vegetation from increased flow may enhance conditions for botulism described by the sludge-bed hypothesis (Enright 1971). High, irregular flows that empty into large, flow-through wetlands, such as on BRMBR, would produce the fluctuating water levels described above. Flooding of dry areas or mudflats due to fluctuating water levels may produce large numbers of invertebrate carcasses (Hunter 1970, Duncan and Jensen 1976), a major requirement for outbreaks under the micro-environment hypothesis.

The sludge-bed hypothesis and micro-environment concept are not mutually exclusive, and they do not exclude other possible mechanisms for outbreak initiation. Neither Poole (1950), who manipulated water levels in mesocosms at BRMBR, nor Moulton et al. (1976), who killed invertebrates with an insecticide in a sewage lagoon, produced botulism in ducks confined to study areas. Moulton et al. (1976) concluded that lack of expected mortality may have been due to lack of concurrent suppression of microorganisms that compete ecologically with *C. botulinum* for organic substrate or other life-history needs. Thus, additional disturbance was necessary for outbreak initiation. The high precipitation and streamflows we associated with outbreaks on BRMBR may represent major disturbances to the microbial ecology of wetland sediments. Such ecological disturbances may improve conditions for *C. botulinum* (Rocke et al. 1999). For example, high precipitation and streamflows increase organic matter deposition and turbidity (J. Kadlec, unpublished data). Temporary superabundance of organic substrate may release *C. botulinum* from prohibitive competition (*sensu* Moulton et al. 1976) long enough for population growth and toxin production to occur.

Management implications

Maintaining stable water levels may be difficult in systems like BRMBR because of variable annual weather patterns and streamflow and the large, flow-through nature of the wetlands. To reduce the risk of uncontrolled water-level fluctuations associated with outbreaks in these types of systems, canals should be used to divert water from riverine sources around management units, instead of through the managed wetlands. Unit size should be decreased to facilitate water-level management and reduce effects of wave action and wind tides. This design would allow rapid water-level manipulation to completely submerge or dry mudflats in the event of an outbreak and thus discourage bird use. The ability to respond quickly to outbreak detection may eliminate the need to permanently inundate important mudflat habitats as recommended by Wobeser (1987). However, careful monitoring for signs of botulism and availability of water for management in a semi-arid environment would be essential for the success of this strategy.

Maintaining stable, deep water levels (Wobeser 1987) would eliminate a habitat type essential for management of local bird populations. Establishing and maintaining wet mudflats for migrating shorebirds and waterfowl is considered a habitat management priority on BRMBR (USFWS 1997) and other similar systems. Managing of habitats for shorebirds requires availability of mudflats and shallow water areas as feeding and loafing sites (Tacha et al. 1994). Additionally, northern pintails and green-winged teal, 2 of the most numerous waterfowl species in this system (USFWS, unpublished report), prefer to feed in mudflats and shallow-water areas (Bellrose 1980).

Wetland managers responsible for habitat management in areas similar in hydrology and botulism outbreak risk should monitor winter–spring

precipitation amounts to determine the relative risk of an outbreak each year. This type of planning effort could aid managers with budgeting of personnel and equipment resources when expensive and time-consuming carcass cleanup efforts are added to normal management activities. Managers also should maintain long-term mortality, precipitation, and flow records to help isolate environmental stimuli that may interact with other ecological factors in each system to trigger avian botulism outbreaks.

Our study of botulism outbreaks on BRMBR demonstrates that historical and local environmental and habitat conditions can be used to better understand and predict local phenomena. This approach may be especially useful to investigate cases such as avian botulism, where average habitat conditions or conditions at a particular site have not previously been useful to predict the same phenomena at other sites (Rocke et al. 1999).

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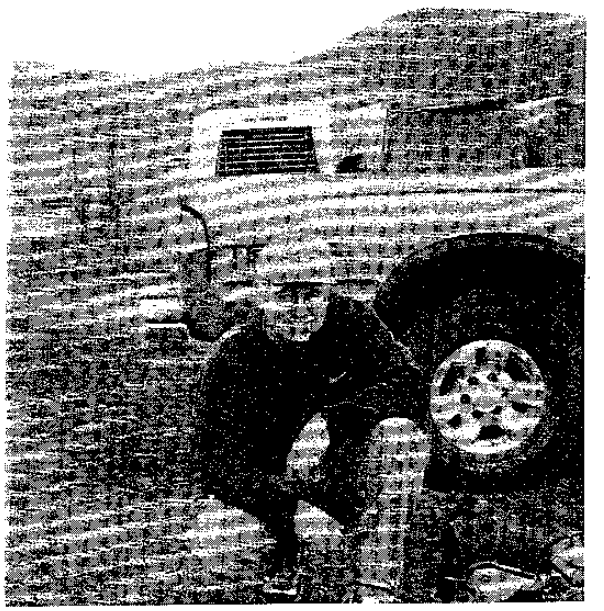

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